2.   A method as claimed in Claim 1 wherein any of the crosses employ preserved gametes.

3.   A method as claimed in Claim 1 wherein the F_1 progeny and some of the N2 progeny exhibit an extreme outlying phenotype.

4.   A method as claimed in Claim 3 wherein the segregating mutation is a heterozygous modifier of the index phenotype selected from a group consisting of an enhancing modifier and a suppressing modifier.

5.   A method as claimed in Claim 1 wherein the dominant allele is a *Min* allele at an *Apc* locus in a mouse.

6.   A method as claimed in Claim 1 wherein the index inbred strain and the founder inbred strain share an isogenic genetic background.

7.   A method as claimed in Claim 6 further comprising the step of mapping the segregating mutation using a mapping partner strain produced by the steps of:
    treating an animal of an index strain with a mutagenic agent to induce point mutations in the treated animal;
    crossing the treated animal to an animal of the index strain to produce F1 progeny; and
    sib-mating F1 and subsequent generation progeny until detrimental and lethal mutations are eliminated.

8.   A method as claimed in Claim 1 wherein the founder inbred mouse strain is produced by a method comprising the step of treating a wild-type inbred mouse with a mutagenic agent to induce point mutations.

9.   A method as claimed in Claim 8 wherein the mutagenic agent is ethylnitrosourea.

26.   A method as claimed in Claim 6 wherein the method identifies a segregating mutation at a genetic locus that modifies tumor multiplicity in a C57BL/6 mouse congenic for the *Min* allele at the Apc locus, the method comprising the steps of:

outcrossing at least one male C57BL/6 mouse carrying random point mutations to a female C57BL/6 mouse congenic for the *Min* allele at the Apc locus to obtain F1 progeny, wherein at least one of the F1 progeny carries both the *Min* allele and a random point mutation; and

backcrossing gametes from male F1 progeny to at least one female C57BL/6 mouse congenic for the *Min* allele at the Apc locus to obtain N2 backcross progeny, wherein at least one of the N2 backcross progeny carries the *Min* allele and has a tumor multiplicity that is modified relative to tumor multiplicity in a C57BL/6 mouse congenic for the *Min* allele at the Apc locus, the modified tumor multiplicity being characteristic of the segregating mutation.

27.   A method as claimed in Claim 26 wherein the modified tumor multiplicity is evaluated according to a method comprising the steps of:

repeatedly applying for random permutations of mice among N2 backcross subkindreds a likelihood ratio test of the null hypothesis that no multiplicity modifier is segregating to obtain a p-value, wherein a p-value of less than 0.05 indicates a potential carrier of the segregating mutation;

when the p-value is less than 0.05, calculating, for each potential carrier that has offspring with information about tumor multiplicity, a LOD score for presence of the segregating mutation, wherein the LOD score is $\log_{10}$ of a ratio of the probability of offspring phenotype data if the potential carrier mouse carries a multiplicity modifier to the probability of offspring phenotype data if the potential carrier mouse does not carry a multiplicity modifier, and wherein the denominator probabilities are calculated from an estimated background distribution and the numerator probabilities are calculated from a mixture of the estimated background distribution and an estimated modified distribution, where the estimated distributions are obtained by the method of maximum likelihood; and

mapping LOD scores of the potential carriers, whereby animals having the highest LOD scores are likely carriers of the segregating mutation.

28.   A method as claimed in claim 26, further comprising the step of mapping the segregating mutation in the N2 backcross progeny using a mapping partner strain.

29.   A method as claimed in Claim 28 wherein the mapping partner strain is produced by the steps of:

treating a C57BL/6 mouse with a mutagen to introduce random point mutations;

crossing the treated mouse to a C57BL/6 mouse to produce F1 progeny; and

sib-mating F1 and subsequent generation progeny until detrimental and lethal mutations are eliminated.